

Research Review: Neuropsychological test performance in pediatric obsessive–compulsive disorder – a meta-analysis

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Background: Research into the neuropsychology of pediatric obsessive–compulsive disorder (OCD) reveals inconsistent results, limiting the ability to draw conclusions about possible neurocognitive deficits in youth with OCD. The aim of this study was to conduct a meta-analysis of the available literature. **Methods:** We identified 36 studies, of which 11 studies met inclusion criteria. Results were categorized into nine functional subdomains: planning, response inhibition/interference control, set shifting/cognitive flexibility, verbal memory, nonverbal memory, processing speed, working memory, visuospatial functions, and attention. For each domain, weighted pooled Hedges' *g* effect size was calculated using random model analyses. **Results:** Small effect sizes were found across all subdomains, none of which were found to be statistically significant. **Discussion:** Results indicate that youth with OCD do not exhibit noteworthy neuropsychological deficits. This is in line with recent suggestions that OCD may not be characterized by clinically meaningful neuropsychological impairments. However, the small number of available controlled studies highlights the urgent need for more neuropsychological research in this population, as well as for further exploration of the neurodevelopmental hypothesis in pediatric OCD. Finally, the relatively low persistence rates of OCD into adulthood should be taken under consideration, especially in the context of the putative neuropsychological performance differences between adult and pediatric OCD populations. **Keywords:** Obsessive–compulsive disorder, pediatric, neuropsychology, meta-analysis, executive function, developmental, cognitive functions.

Introduction

Obsessive–compulsive disorder (OCD) is a burdensome condition characterized predominantly by obsessions – intrusive and unwanted thoughts and urges that cause distress and anxiety – and compulsions, which are repetitive mental or behavioral rituals that the individual feels driven to perform, usually reducing or preventing the distress that accompanies obsessions (American Psychiatric Association, 2013). The worldwide prevalence of OCD ranges between 1.5 and 3% (Okasha et al., 2000; Ruscio, Stein, Chiu, & Kessler, 2010). As nearly 50% of adult patients with OCD exhibit symptoms that can be traced to childhood, a bimodal distribution model of OCD onset has been proposed that suggests one peak in preadolescents and a second in early adulthood (Geller, 2006).

In the past 25 years, a vast body of knowledge has developed from a growing interest in neurobiological mechanisms and neuropsychological functioning in OCD, alongside research into phenomenology, pharmacotherapy, and psychotherapy. This body of knowledge has yielded the cortico-striato-thalamo-cortical (CSTC) neurobiological model of OCD that

hypothesizes a frontostriatal pathophysiology (Milad & Rauch, 2012; Saxena & Rauch, 2000). However, in contrast to the relatively consistent results seen in resting-state imaging studies, adult OCD neuropsychological studies are notoriously divergent (Kuelz, Hohagen, & Voderholzer, 2004). In fact, in a recent meta-analysis examining neuropsychological test performance in adult patients with OCD, Abramovitch, Abramowitz, and Mittelman (2013) significant heterogeneity was found across most neuropsychological domains.

Nevertheless, the adult OCD literature indicates underperformance on neuropsychological tests especially in nonverbal memory, planning, and processing speed. Evidence for underperformance on set-shifting tasks, as well as response inhibition, visuospatial functions, and visuospatial working memory, is less conclusive and appears to be contingent upon task selection and the degree of the test's complexity/cognitive load (Abramovitch & Cooperman, 2015). For example, the majority of adult OCD studies utilizing commission errors on Go-No/Go (GNG) tests and Continuous Performance Tests (CPTs) as an indicator for response inhibition reveal comparable performance to healthy controls (Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Krishna et al., 2011; Gonsalvez, & Johnstone,

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2014). On the other hand, most studies assessing response inhibition using the stop signal task (SST) indicate deficient performance among OCD samples (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; De Wit et al., 2012; Penades et al., 2007). A similar difference is revealed in studies utilizing the copy trial of the Rey-Osterrieth Complex Figure (ROCF) test versus the Wechsler Adult Intelligence Scale (WAIS) Block Design test to assess visuospatial functions – where most studies indicate intact performance on the former (Kim, Park, Shin, & Kwon, 2002; Roth, Baribeau, Milovan, & O'connor, 2004) and deficient performance on the latter (Moritz, Hottenrott, Jelinek, Brooks, & Scheurich, 2012; Tukel et al., 2012). Overall, it has been suggested that although a large body of adult neuropsychological literature is available, a disorder-specific neuropsychological profile in OCD could not yet be determined (Abramovitch & Cooperman, 2015; Abramovitch et al., 2013; Kuelz et al., 2004). Moreover, it has been suggested that this body of literature does not support the presence of clinically meaningful neuropsychological impairments in adult OCD (Abramovitch et al., 2013; Ahmari, Eich, Cebenoyan, Smith, & Blair Simpson, 2014; Simpson et al., 2006).

Relative to the numerous adult studies, considerably less neuropsychological research has been conducted in pediatric OCD. Moreover, the existing studies have yielded inconsistent results, with children affected by OCD sometimes outperforming nonpsychiatric (healthy) children, and vice versa, depending on domain and type of neuropsychological test (Andres et al., 2007; Beers et al., 1999). To account for these inconsistencies, some authors have proposed that factors such as age of onset or comorbidity may mediate different neuropsychological performance in pediatric OCD (Andres et al., 2007; Ornstein, Arnold, Manassis, Mendlowitz, & Schachar, 2010). Others have concluded that youth with OCD do not exhibit neuropsychological impairment in early stages of the disorder (Beers et al., 1999). Nevertheless, the only available (narrative) review of this literature concluded that the considerable variability in findings across studies prevents firm conclusions regarding neuropsychological functioning in children with OCD (Abramovitch, Mittelman, Henin, & Geller, 2012).

To our knowledge, no quantitative or meta-analytic reviews of the pediatric OCD neuropsychology literature are available. Thus, the primary aim of this study was to use meta-analytic methodology to assess the magnitude of differences between youth with OCD and nonpsychiatric (control) children on neuropsychological test performance. In line with the recent meta-analysis on adult neuropsychological functions in OCD (Abramovitch et al., 2013), we focused on six primary neuropsychological domains (i.e., attention, executive functions, memory, processing speed, visuospatial abilities, and working

memory) and a total of 10 subdomains (i.e., sustained attention, planning, response inhibition/interference control, set shifting/cognitive flexibility, verbal memory, nonverbal memory, processing speed, visuospatial abilities, verbal working memory and visuospatial working memory). Nontraditional neuropsychological tasks (e.g., decision-making tasks), as well as outcome measures that are not considered a part of the original test (e.g., organizational scores on the ROCF test), were excluded.

Method

Retrieval and selection of studies

This study employed a three-stage searching strategy. First, we conducted a search using the ISI Web of Knowledge, MEDLINE, and PsycINFO electronic databases up to April 2014, using the search terms 'OCD', 'obsessive-compulsive disorder', and 'pediatric', which were cross-referenced with the terms 'neuropsych*', 'neurocog*', 'memory', 'executive function', 'processing speed', 'visuospatial', 'attention', 'working memory', 'response inhibition', 'planning', and 'set shifting'. In the second stage, we examined publications' reference lists. The third stage consisted of solicitation of unpublished data from researchers in the field of neuropsychology of OCD. As depicted in Figure 1, this search produced 36 research articles. In further designating research as appropriate for inclusion in the meta-analysis, only controlled studies were considered. That is, we included a study only if it contained at least one comparison between a group of DSM-diagnosed pediatric (17 years old or younger) patients with OCD and a healthy (i.e., screened for the absence of a lifetime psychiatric or neurologic diagnosis) control group using one or more neuropsychological tests.

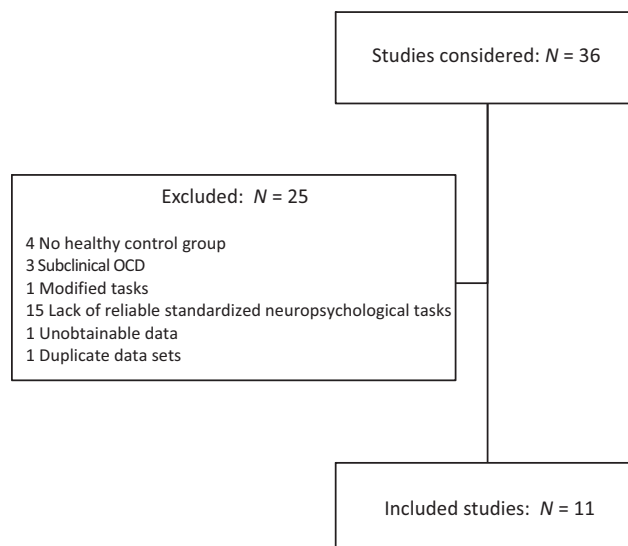


Figure 1 Study selection process for the meta-analysis of neuropsychological investigations in pediatric obsessive-compulsive disorder (OCD)

We excluded any study that did not include healthy controls and instead reported only comparisons between multiple patient groups (e.g., patients with OCD vs. patients with schizophrenia). Of the 36 studies identified, 32 met these initial criteria (i.e., including healthy control samples).

Of the 32 studies, three studies were excluded because they examined remitted or subclinical samples. One study was excluded due to modification of a standardized neuropsychological task. Fifteen additional studies were excluded due to lack of reliable standardized neuropsychological tasks. We also excluded one study because it failed to provide sufficient data necessary to calculate or estimate effect sizes (data were subsequently unobtainable from the authors). Finally, one study was excluded because it used a dataset already included in a study analyzed in the present meta-analysis. As shown in Figure 1, these exclusions yielded a final count of 11 studies ($N_{\text{OCD}} = 227$, $N_{\text{controls}} = 242$; Andres et al., 2007; Shin et al., 2008; Taner, Emel, & Oner, 2011; Beers et al., 1999; Behar et al., 1984; Chang, Mccracken, & Piacentini, 2007; Gruner et al., 2012; Huyser, Veltman, Wolters, De Haan, & Boer, 2010; Kodaira et al., 2012; Ornstein et al., 2010; Zandt, Prior, & Kyrios, 2009), the year of publication ranging from 1984 to 2012. Analyses were conducted using Microsoft Excel in accordance with standard meta-analytic formulae (Borenstein, Hedges, Higgins, & Rothstein, 2011).

Variables recorded and coded from studies

The following general information was recorded from the 11 studies: (a) publication status, (b) publication year, and (c) country where the research was conducted. The following participant characteristics of each study were also recorded: (a) sample size of OCD and control groups, (b) mean age, (c) mean age of OCD onset, (d) years of education, (e) percent of males in the OCD group, (f) mean scores on measures of OCD severity (e.g., the Children's Yale-Brown Obsessive-Compulsive Scale), and (g) percent of patients with OCD receiving SRI or neuroleptic (i.e., antipsychotic) medication. Table 1 summarizes the demographic and clinical characteristics of each of the 11 studies. As can be seen, a notable range was observed in sample sizes ($M_{\text{OCD}} = 20.6$, $SD = 5.8$, range = 14–35), as well as gender composition (% males, $M_{\text{OCD}} = 53.3\%$, $SD = 15.3$, range 29–82%). On average, the OCD sample had moderate to severe OCD symptoms. Five of the studies were conducted in the United States, with one study each in South Korea, Spain, Australia, Japan, the Netherlands, and Turkey. Finally, in five studies, the OCD group was unmedicated.

We recorded the specific neuropsychological tests administered in each study in addition to the domain of functioning (e.g., memory) that each test evaluated. We also recorded the subdomain of functioning

measured by each test (e.g., verbal memory, nonverbal memory). Table 2 lists the domains, subdomains, and tests coded from all studies in the meta-analysis. In certain cases, some but not all of the neuropsychological outcome measures were uncommon. In these cases (e.g., the TMB-TMA index score), we documented only the conventional variables. Finally, from each study, we recorded (a) the number of tests ($M = 8.6$, $SD = 5.36$, range = 1–20) and (b) the number of testing sessions ($M = 1.33$, $SD = 0.52$, range = 1–2).

Effect size computation

Due to the small sample sizes, we expressed the results for all tests in all studies in terms of Hedges' g (Hedges & Olkin, 1982), a standardized measure of effect size that was calculated by subtracting the control group's mean from the OCD group's mean and dividing by the pooled standard deviation. Therefore, a specific effect size was generated for each test on which an OCD group was compared with a control group. Positive effect sizes (e.g., $g = 0.25$) indicated better neuropsychological functioning in the OCD group compared to the control group, while negative effect sizes (e.g., $g = -0.50$) indicated the opposite. Cohen (1977) suggested that small, medium, and large effect size magnitudes correspond to 0.2, 0.5, and 0.8, respectively.

Effect sizes were calculated using the sample size, means, and standard deviations reported in each study; this information was available or obtainable from authors in all instances. We computed effect sizes only after information from all studies had been coded so as to reduce the potential for knowledge of a study's results to bias the coding of its characteristics. We used a random effects model and assumed heterogeneity across k samples because, as previously mentioned, pediatric OCD neuropsychological studies have historically yielded inconsistent and somewhat conflicting results. That is, we assumed that these incongruities would yield effect size heterogeneity across studies, in contrast to a common effect size across studies as in the case of the fixed effect model. To determine whether effect sizes were consistent across comparisons, we calculated a homogeneity statistic Q , which had an approximate chi-square distribution with $k-1$ df , where k is equal to the number of effect sizes. We also computed τ^2 and I^2 coefficients.

Preliminary analyses

Within each domain and subdomain of neuropsychological functioning, the performance of OCD and control groups was often assessed by multiple tests within a specific study. Therefore, we determined a mean effect size for each domain and subdomain within each study using the available test results as listed in Table 3. This procedure yielded 37 domain

Table 1 Demographics and selected methodological characteristics of the studies included in the meta-analysis ($N = 11$)

Study (year)	DSM	OCD, <i>n</i>	HC, <i>n</i>	<i>M</i> age (OCD)	<i>M</i> age (HC)	% OCD male	OCD severity	% Medicated	Comorbidity, <i>n</i>
Behar et al. (1984)	III	17	16	13.7	14.0	82	Leyton; unavailable	0%	3 MDD
Beers et al. (1999)	III	21	21	12.3	12.2	57	CYBOCS; Mdn obsessions = 10; Mdn compulsions = 11	0%	0
Andres et al. (2007)	IV-TR	35	35	13.8	13.8	51	CYBOCS total = 26.8	13/35 SSRI	0
Chang et al. (2007)	IV	16	15	12.6	11.9	69	CYBOCS total = 20.8	1/16 SSRI	4 MDD, 6 GAD
Shin et al. (2008)	IV	17	23	12.1	10.1	65	Leyton total = 32.7	6/17 SSRI ^a	0
Zandt et al. (2009)	IV-TR	17	18	12.3	11.9	47	N/A	4/17 SSRI ^b	0
Huysen et al. (2010)	IV	25	25	13.9	13.7	36	CYBOCS total = 24.9	0%	4 SAD, 16 SPH, 16 SpPh, 8 GAD, 4 PTSD, 8 Dep, 4 Dysth, 8 ADHD, 4 ODD, 8 Tic
Ornstein et al. (2010)	IV	14	14	12.9	12.8	29	CYBOCS total = 19.3	3/14 SSRI	2 TS, 2 GAD
Taner et al. (2011)		20	30	11.6	11.3	40	N/A	0%	0
Gruner et al. (2012)		23	23	14.3	14.2	56	CYBOCS total = 26.9	12/23 SSRI	4 MDD, 2 PD, 2 SPH, 5 ADHD
Kodaira et al. (2012)	IV-TR	22	22	13.6	13.5	54	CYBOCS total = 22.4	9/22 SSRI, 1/22 CPM	2 Tic, 3 TTM, 1 Stu, 1 Som

DSM, diagnostic and statistical manual of mental disorders; OCD, obsessive-compulsive disorder; HC, healthy controls; *M* age, mean age in years; Leyton, Leyton Obsessional Inventory; CYBOCS, Children's Yale-Brown Obsessive-Compulsive Scale; Mdn, median; N/A, not available; SSRI, selective serotonin reuptake inhibitor; CMP, clomipramine; MDD, major depressive disorder; GAD, generalized anxiety disorder; SAD, separation anxiety disorder; SPH, social phobia; SpPh, specific phobia; PTSD, post-traumatic stress disorder; Dep, depression; Dysth, dysthymia; ADHD, attention deficit/hyperactivity disorder; ODD, oppositional defiant disorder; Tic, tic disorder; TS, Tourette's syndrome; PD, panic disorder; TTM, trichotillomania; Stu, stuttering; Som, somatization disorder.

^aOf the six medicated participants, one or more were on risperidone (1 mg; exact information unobtainable).

^bThe authors reported that additional two participants were on unknown medication.

Table 2 Neuropsychological domains, subdomains, and outcome measures

Domain	Subdomain	Tests
Attention	(a) Sustained attention	CPT (omission errors)
Executive Functions	(a) Planning	TOL, TOH
	(b) Response inhibition/interference control	CPT (commission errors), Stop Task RT, Stroop interference
Memory	(c) Set shifting/cognitive flexibility	Design fluency, verbal fluency, TMB, WISC similarities, WCST
	(a) Verbal memory	CVLT, WMS Logical Memory
	(b) Nonverbal memory	ROCF
Processing speed	(a) Processing speed	CPT RT, Stop Task RT, Stroop (Congruent trial RT), TMA, WISC Digit Symbol, WISC Coding, WISC Symbol Search
Visuospatial abilities	(a) Visuospatial abilities	ROCF Copy, WISC Block Design
Working memory	(a) Working memory	WISC Digit Span, WISC Arithmetic
	(b) Spatial working memory	SWM

CPT, Continuous Performance Test; TOH, Tower of Hanoi; TOL, Tower of London; TMB, Trail Making Test, Part B; WISC, Wechsler Intelligence Scale for Children; WCST, Wisconsin Card Sorting Test; CVLT, California Verbal Learning Test; WMS, Wechsler Memory Scale; ROCF, Rey-Osterrieth Complex Figure; RT, reaction time; TMA, Trail Making Test, Part A; SWM, spatial working memory.

effect sizes and 47 subdomain effect sizes and allowed for separate analyses at the domain and subdomain levels. Effect sizes derived from multiple tests within a given study are likely to be less variable than effect sizes drawn from different studies (Robinson, Berman, & Neimeyer, 1990). To eliminate this potential for nonindependence of observations, we aggregated our data by averaging effect sizes derived from each study within domains (Hedges, 2007). Thus, each study contributed only a single effect size in each analysis, retaining separate

effect sizes for each domain and subdomain, so that these could be examined in separate analyses at the domain and subdomain level.

Results

Table 3 presents the individual aggregated effect sizes for each neuropsychological subdomain derived from each study. A summary of the mean effect sizes for the domains and subdomains of neuropsychological functioning is presented in

Table 3 Effect sizes (Hedges' *g*) for neuropsychological subdomains across studies in the meta-analysis.

Study	OCD, <i>n</i>	Neuropsychological subdomain							
		Planning	RI/IC	SS/CF	VM	NVM	PS	VSA	WM
Behar et al.	17					-0.47		-0.38	
Beers et al.	21	0.06	-0.02	0.45	0.53		-0.17	0.15	0.29
Andres et al.	35			-0.31	-0.52	-0.73	-0.20	-0.41	0.10
Chang et al.	16		-0.49		-0.47	0.29	-0.52	0.03	-0.24
Shin et al.	17		0.04	-0.66		-0.19	-0.68	-0.70	-0.12
Zandt et al.	17			-0.15					
Huyser et al.	25	-0.60							
Ornstein et al.	14	-0.70	0.16	-0.01	-0.01	0.34	0.04	0.19	-0.01
Taner et al.	20		-0.07	-1.08			0.70	-0.73	-0.46
Gruner et al.	23		-0.11	-0.40			-0.15		
Kodaira et al.	22			0.05					

Negative effect sizes indicated reduced performance among OCD samples compared to controls.

RI/IC, response inhibition/interference control; SS/CF, set shifting/cognitive flexibility; VM, verbal memory; NVM, nonverbal memory; PS, processing speed; VSA, visuospatial abilities; WM, working memory.

Table 4. Because the attention domain was examined in only a single study, only one effect size for this domain could be calculated ($g = -0.52$; Shin et al., 2008). Accordingly, this domain was excluded from subsequent analyses. As can be seen, all mean effect sizes were negative, suggesting that on average, across studies, patients with OCD performed more poorly than did nonpsychiatric control groups. However, all mean effect sizes were small in magnitude, and all effect sizes were not found to be statistically significant. Of the five domains, the largest mean effect size was found on tests of visuospatial abilities, where on average, patients with OCD performed approximately one quarter of a standard deviation worse than control groups. The second largest effect size was found for executive functions, wherein OCD groups scored less than a quarter of a standard deviation below controls on average. Working memory evidenced the smallest effect size, approaching zero. The test of homogeneity (*Q*) for major domains did not yield significant heterogeneity.

Table 4 presents a summary of the mean effect sizes for the subdomains of neuropsychological functioning. As can be seen, all mean effect sizes were negative (i.e., control groups outperformed patients with OCD). However, no effect sizes differed significantly from zero (See Figure 2 for a graphic depiction of effect sizes across domains and subdomains). Even within the same domain, the variability in subdomain effect sizes was noteworthy. For example, within the executive function domain, the mean effect size for planning ($g = -0.40$; the largest subdomain effect size) was substantially larger than that for response inhibition/interference control ($g = -0.07$). Interestingly, the subdomains yielding the smallest effect sizes, nearly approaching zero (i.e., response inhibition/interference control and working memory), were also found to be homogeneous, rendering further support to the lack of differences on tasks tapping these constructs between youth with OCD and controls. Notably, the small number of effect sizes precluded any meaningful statistical analyses to examine possible moderator variables.

Table 4 Weighted mean effect sizes and tests of homogeneity by neuropsychological subdomain for studies included in the meta-analysis

Domain/Subdomain	Studies	<i>N</i> OCD/HC	Effect size				Variability across studies			
			<i>g</i>	Lower CI	Upper CI	<i>p</i>	<i>Q</i>	<i>p</i>	τ^2	I^2
Executive functions	10	210/226	-0.22	-0.46	0.01	.063	13.7	>.10	0.05	34%
Planning	3	60/70	-0.40	-0.87	0.06	.087	3.4	>.10	0.07	41%
Response inhibition/interference control	6	111/126	-0.07	-0.32	0.19	.609	1.9	>.10	0.00	0%
Set shifting/cognitive flexibility	8	169/186	-0.26	-0.56	0.05	.101	14.4	<.05	0.10	10%
Memory	6	120/134	-0.14	-0.50	0.22	.445	9.9	<.10	0.10	50%
Verbal memory	4	86/95	-0.15	-0.64	0.34	.547	7.7	<.10	0.15	15%
Nonverbal memory	5	99/113	-0.18	-0.62	0.25	.404	9.5	<.05	0.14	58%
Processing speed	7	146/161	-0.14	-0.44	0.17	.387	10.8	<.10	0.08	45%
Visuospatial abilities	7	140/154	-0.28	-0.56	0.00	.052	8.5	>.10	0.04	29%
Working memory	6	123/138	-0.04	-0.29	0.20	.730	3.6	>.10	0.00	0%

OCD, obsessive-compulsive disorder; HC, healthy controls; CI, confidence interval.

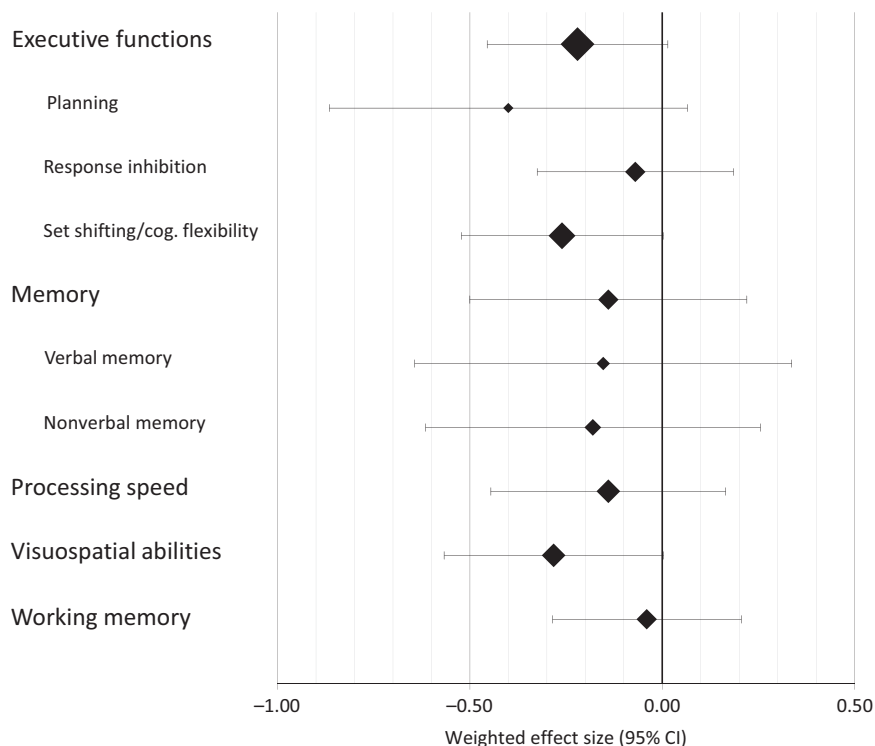


Figure 2 Forest plot of major domains and sub domains' weighted effect sizes and corresponding 95% confidence intervals. 'Diamond' sizes correspond to sample size. Negative effect sizes indicate that patients with OCD underperformed control groups

We examined the possibility that publication bias would inflate the effect sizes, as would be the case if studies with significant findings (large effect sizes) were published, whereas those with null results (small effect sizes) were not. We applied two methods to evaluate publication bias. First, we calculated the fail-safe N (Orwin, 1983) to determine the necessary number of unpublished studies reporting small effect sizes that would be required to reduce our overall mean effect size ($g = -.27$, $p = .004$) to a trivial level. This evaluation suggested that over 12 unpublished studies with effect sizes of 0.00 would be necessary to overturn our findings by decreasing the effect size to a negligible level. If the fail-safe N number exceeds the quantity of already published studies, this is thought to be sufficient evidence to suggest the absence of a significant publication bias. Second, we computed the Egger's regression intercept test and the funnel plot (Egger, Davey Smith, Schneider, & Minder, 1997). Visual inspection of the funnel plot revealed no indication for asymmetry (see online appendix, Figure S1), and the Egger's regression intercept test revealed no significant effect ($t(9) = 0.55$, $p = .30$). Thus, we concluded that a publication bias was unlikely to have affected our results.

Discussion

In contrast to the large body of neuropsychological research on adults with OCD, such investigations with pediatric OCD samples have been scant.

Moreover, the results from available studies in pediatric OCD are highly inconsistent; whereas some researchers have found evidence of neuropsychological impairments in this population, the majority of studies have concluded that only limited deficits (if any) exist in this population. To our knowledge, however, the present study represents the first attempt to meta-analyze this body of literature. The present review included 11 studies spanning three decades, and the resulting mean effect sizes for all neuropsychological domains and subdomains were found to be nonsignificant. However, the lack of statistical significance may be heavily influenced by the small number of included studies (and small samples across studies), and more emphasis should be placed upon interpretation of the magnitude of effect sizes found in the present study. Overall effect sizes for all major domains ranged between -0.04 and 0.28 corresponding to an overall performance difference of zero to one quarter of a standard deviation between youth with OCD and controls. This denotes the presence of only slight differences between patients and controls and also indicates that underperformance of patients with OCD cannot be labeled, at this time, as clinically significant or impaired. This is because a difference of 2.0 standard deviations from healthy individuals' performance is generally considered the norm for identification of clinically significant impairment (Lezak, 2012). However, it is important to note that this does not preclude cases of children with OCD that may present with more

substantial deficits, given that these benchmarks for clinically significant impairments may be more relevant to individuals rather than to groups. Thus, the low omnibus effect size found in the present meta-analysis does not eliminate the possibility that some children with OCD may exhibit neuropsychological deficits.

On the domain level, no significant heterogeneity was found. Significant heterogeneity was found only for the set shifting/cognitive flexibility and nonverbal memory subdomains, providing support to the reliability of our findings that should be taken in the context of the limited sample size. That is, despite the seemingly inconsistent results thought to characterize this body of literature, and the small number of studies included, significant variability was not observed across domains other than processing speed and memory. We conclude that despite the small number of studies, effect sizes between studies are generally very small, with significant heterogeneity found on two subdomains. Of note, studies that employed tasks that are not considered reliable standardized neuropsychological tests (and were subsequently excluded from this study) reveal a similar pattern of results. These studies, employing tasks such as the Simon task, modified Stroop task, or modified set-shifting task, usually report no performance differences between pediatric OCD and control samples, corresponding to small effect sizes (Britton et al., 2010; Hajcak, Franklin, Foa, & Simons, 2008; Ota et al., 2013; Rubia, Cubillo, Woolley, Brammer, & Smith, 2011). Only a few studies deviated from this trend, reporting moderate to large effect sizes (Flament et al., 1988).

Nevertheless, due to the small number of available studies, we were unable to conduct analyses of possible moderators that might account for effect size heterogeneity in these subdomains. Notably, potential moderators, including but not limited to OCD symptom severity, depressive severity, and medication status, were not found to be statistically significant in a recent meta-analysis of neuropsychological test performance in adult OCD (Abramovitch et al., 2013). However, more research is needed to assess the potential moderating effect of such factors in pediatric OCD. Of note, qualitative examination of the included studies yielded no overall notable differences between unmedicated and medicated samples of youth with OCD. In fact, Andres et al. (2007) compared a subsample of 16 medicated youth with OCD with 19 unmedicated patients and found no significant difference between the groups on a comprehensive neuropsychological battery.

Our results appear to be in contrast to the prevailing neurobiological models of OCD that predict neuropsychological deficits, particularly in executive functions. However, there are some indications that neurobiological processes in youth with

OCD (especially in younger children) may be different and more dynamic than in adults, given typical neurodevelopmental processes occurring in childhood. For example, in contrast to the vast majority of resting-state functional connectivity studies in adult OCD that repeatedly demonstrate abnormally *increased* connectivity (for a recent review, see Pauls, Abramovitch, Rauch, & Geller, 2014), there are indications that young children with OCD may display *decreased* connectivity at rest (Fitzgerald et al., 2011). In fact, Rosenberg and Keshavan (1998) proposed that pediatric OCD may be characterized by deficient neuromaturational processes such as pruning and myelination (Rosenberg & Keshavan, 1998). However, to our knowledge, this OCD-specific neurodevelopmental hypothesis was not explored further (for a review, see Abramovitch et al., 2012).

To our knowledge, two hypothetical explanations were offered that may account for the lack of meaningful effect sizes for cognitive underperformance reported in the present study. One possibility is that cognitive deficits in OCD become significant only toward young adulthood. In their study of neuropsychological performance in pediatric OCD, Beers et al. (1999) concluded that 'These results suggest that children with OCD do not exhibit clinically significant cognitive deficits early in the illness. Disturbance of cognitive function may become significant over time' (Beers et al., 1999). This speculative hypothesis, which may correspond to the neurodevelopmental hypothesis of pediatric OCD (Rosenberg & Keshavan, 1998), did not receive further research support. However, in the context of a neurodevelopmental framework, it is important to consider that depending on the definition of remission, nearly 60% of children experience remission of OCD in adulthood (Stewart et al., 2004). Thus, assuming that OCD is associated with underperformance on neuropsychological tests, it would be reasonable to expect larger effect sizes in adults with OCD compared to pediatric samples. However, this speculative hypothesis requires empirical investigation utilizing a comprehensive battery of neuropsychological tests as part of a longitudinal study.

A second explanation offered by Abramovitch et al. (2013) argues that small to moderate effect sizes corresponding to cognitive underperformance do not represent clinically significant impairment. The authors' argument – grounded in cognitive and clinical neuropsychology conventions – led to their conclusion that 'although the present meta-analytic examination offers evidence for poorer performance in OCD relative to healthy individuals across the majority of neuropsychological domains, these differences may not be clinically meaningful' (Abramovitch et al., 2013). With the limitation of the small number of studies included in the present meta-analysis, our results may be in support of the latter

argument. However, further research would be instrumental in clarifying this complex question.

Notably, the present study indicates that aside from tasks assessing response inhibition and working memory, children with OCD tend to underperform to a small degree on tests assessing the majority of other neuropsychological constructs. Hypothetically, this could be a nonspecific small reduction in general cognitive abilities attributed to the P factor (i.e., having a psychopathology; Caspi et al., 2013), but more research is required to assess the specificity (or lack thereof) of these findings. In terms of clinical implications, there is an overwhelming lack of research in both adult and pediatric OCD exploring the real life sequelae of neuropsychological underperformance in OCD. We would encourage researchers to assess ecologically valid functional correlates of neuropsychological test performance in pediatric OCD, and suggest that clinicians gauge them as part of their evaluation when cognitive deficits are reported. It would also be useful to assess motivation as part of the administration of neuropsychological batteries, due to the possibility that children with OCD may be able to invest more efforts in neuropsychological assessments. That is, it is possible, albeit speculative, that adults with OCD have been struggling with the disorder for much longer and hence may be less able to invest efforts when tested.

The present findings pertaining to youth with OCD are somewhat smaller in magnitude compared to the ones obtained in recent meta-analyses of neuropsychological functioning in adult patients with OCD (Abramovitch et al., 2013; Shin, Lee, Kim, & Kwon, 2014). These studies reported that on average, adults with OCD exhibited underperformance across neuropsychological domains that were of medium effect sizes. For example, effect sizes for nonverbal memory in adult OCD range between .74 and .76, compared to .18 for the pediatric sample assessed in the present study. Given the paucity of research on neuropsychological test performance in pediatric OCD, as well as the lack of longitudinal neuropsychological studies and paucity of neuromaturation investigations in this population, it is difficult to offer a cogent hypothesis as to this difference – which does not appear to exist in other psychiatric disorders such as ADHD and depression (Frazier, Demaree, & Youngstrom, 2004; Snyder, 2013; Wagner, Muller, Helmreich, Huss, & Tadic, 2015). However, the body of pediatric neuropsychological research in these disorders is substantially larger than in OCD.

Perhaps the most prominent difference between the results of our study and other neuropsychological meta-analyses in adult OCD (Abramovitch et al., 2013; Shin et al., 2014; Snyder, Kaiser, Warren, & Heller, 2015) lies in the extent of the effect sizes for response inhibition. As opposed to adults where effect sizes for response inhibition underperformance are of medium magnitude (.49 to .55), the

present study indicates that in pediatric OCD, the effect size for response inhibition is approaching zero (.07). This may be important due to suggestions made in the recent decade that response inhibition may be a candidate endophenotype of OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). One hypothetical explanation may lie in task selection. As noted above, in their critical review of the adult OCD neuropsychological literature, Abramovitch and Cooperman (2015) note that the literature indicates performance differences across the three major neuropsychological paradigms of response inhibition. The authors indicate that the vast majority of studies utilizing the GNG and CPT paradigms reveal comparable performance between adult OCD and control samples, where only a minority of studies employing SST indicate comparable performance. In addition, studies using the Stroop test are inconsistent (Abramovitch & Cooperman, 2015). It has been suggested that the three paradigms assess different subconstructs of response inhibition. That is, the CPT and GNG tests assess *action suppression*, the SST assesses *action cancellation*, and the Stroop test assesses *interference control*. Moreover, there are indications of different neural substrates associated with the three paradigms (Eagle, Bari, & Robbins, 2008). In the present meta-analysis, only one included study employed the SST, while the majority of studies employed the Stroop test and the CPT. Thus, task selection for the assessment of response inhibition in pediatric OCD may explain the apparent performance difference between children and adults, and researchers are encouraged to utilize the SST in future studies, and ideally employ tests associated with the three paradigms within each study.

Conclusion

The present meta-analysis offers evidence for comparable neuropsychological performance in youth with OCD compared to controls, where all effect sizes were found to be small. Results indicate an overall small degree of underperformance on most subdomains, intact performance on response inhibition and interference control subdomains, and a small to moderate degree of underperformance in the subdomain of planning. It is important, however, to consider the small number of studies included, which highlights the urgent need for more research tapping neuropsychological functions in pediatric OCD. Moreover, we note that more than half of individuals diagnosed with OCD in childhood experience remission in adulthood (Stewart et al., 2004), which may account for the differences found between the two populations on neuropsychological test performance. However, this hypothesis requires further empirical longitudinal research focusing upon neurodevelopmental, and particularly neuro-maturation, processes.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Figure S1. Funnel plot of standard error by Hedge's *g*.

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Key points

- Neuropsychological investigations in pediatric obsessive-compulsive (OCD) yield inconsistent results.
- We conducted the first meta-analysis of cognitive functions in pediatric OCD.
- Small and statistically insignificant effect sizes were found across domains.
- Our results suggest that youth with OCD do not exhibit meaningful neuropsychological deficits.
- Given the small number of available controlled neuropsychological studies in pediatric OCD, more research using larger samples is highly needed in this population.

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